Amendments to the Specification:

Please insert the paper copy of the Sequence Listing filed herewith following the Oath/Declaration.

Please replace the paragraph beginning at page 1, paragraph 12 with the following amended paragraph:

HRGP is a heparin-binding plasma protein identified by Heimburger at al. See, Heimburger et al. (1972) Physiol. Chem. 353:1133-1140. The average concentration of HRGP in plasma is around 100 μg/ml. See, Drasin and Sahud (1996) Thrombosis Research 84:179-188. The amino acid sequences of mouse, rat, (Hulett and Parish (2000) Immunology and Cell Biology 78: 280-287), rabbit (Borza et al. (1996) Biochemistry 35:1925-1934) and human (Koide et al. (1986) Biochemistry 25:2220-2225) HRGP have been resolved. Structurally, the HRGP molecule can be divided into three main domains. The amino-terminal domain, encompassing about 250 amino acid residues, contains two cysteine protease inhibitor (cystatin)-like stretches, which allows the classification of HRGP as a member of a cystatin superfamily together with α2HS glycoprotein and kininogen. There are six putative sites for N-linked glycosylation in the amino terminal portion of HRGP. A central domain contains tandem repeats of the pentapeptide [H/P]-[H/P]PHG (SEQ ID NO:1). Both the central domain and the 105 amino acid C-terminal domain are di-sulfide bonded to the cystatin-like stretches in the amino-terminal domain (Borza et al., 1996). HRGP binds heparin/heparan sulfates (Lijnen et al. (1983) J. Biol. Chem. 258:3803-3808) in a pH-dependent interaction (Peterson et al. (1987) J. Biol. Chem. 262: 7567-7574). The isolated histidine-proline-rich middle domain mediates heparin binding (Borza et al., 1996), but the amino terminal domain has also been implicated in heparin binding. See, Koide et al. (1986) FEBS Lett. 194:242-244. A congenital deficiency of HRGP has been mapped to a single-nucleotide mutation, which results in replacement of Gly85 to Glu in HRGP. This mutation leads to inefficient processing of the protein, the majority of which is retained intracellularly. As a consequence, the serum levels of HRGP are reduced to 25-30% of normal levels. See, Shigekiyo et al. (1993) Thromb. Haemost. 70:263-265 and Shigekiyo et al. (1998) Blood 91:128-133.

Please replace the paragraph beginning at page 2, paragraph 25 with the following amended paragraph:

In one aspect, the invention features a pharmaceutical composition including an HRGP polypeptide, and an anti-angiogenic agent or an anti-neoplastic agent. The composition can include a pharmaceutical carrier acceptable for administration to a mammal. The HRGP polypeptide can include, for example, intact HRGP, a modified HRGP polypeptide or a fragment of intact HRGP. This fragment of intact HRGP may include, but is not limited to, the amino terminal domain, the central domain or the C-terminal domain of intact HRGP, or a fragment including at least one tandem repeat of the pentapeptide [H/P]-[H/P]PHG (SEQ ID NO:1). The anti-angiogeneic agent may be, for example, angiostatin, thrombostatin, endostatin, interferon-I, interferon-inducible factor 10, platelet factor 4 or a COX-2 inhibitor.

Please replace the paragraph beginning at page 8, paragraph 6 with the following amended paragraph:

The term HRGP polypeptide includes polypeptides that contain one of the three main domains of the HRGP molecule, and functional fragments thereof. The amino-terminal domain, encompassing about 250 amino acid residues, contains two cysteine protease inhibitor (cystatin)-like stretches. There are six putative sites for N-linked glycosylation in the amino terminal domain of HRGP. The central domain contains tandem repeats of the pentapeptide [H/P]-[H/P]PHG (SEQ ID NO:1) and the C-terminal domain contains 105 amino acids. Both the central domain and the C-terminal domain are di-sulfide bonded to the cystatin-like stretches in the amino-terminal domain (See Borza et al., 1996). The isolated histidine-proline-rich middle domain mediates pH-dependent heparin binding (Borza et al., 1996). The amino terminal domain has also been implicated in heparin binding.